Physiological Role of Dietary Sodium in Human Health and Implications of Sodium Reduction in Muscle Foods

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□ SODIUM CHLORIDE consumption has become a major issue in the food industry, primarily because the sodium ion seems closely linked to human hypertension (Freis, 1976; Altschul and Grommet, 1980) and because the average consumption of sodium is 10–20 times that necessary for physiologial balance (NAS, 1980). Public awareness of sodium is becoming evident in consumer surveys. A study recently completed by the National Pork Producers Council showed consumer concern for sodium to be nearly twice as great in 1982 as in 1981 (Anon., 1983b), and over 70% of consumers view salt as a potential health threat (Andres, 1982). While the sodium-hypertension relationship is not one of simple cause-and-effect, most sources agree that sodium intake probably should be reduced. Muscle foods (red meat, fish, and poultry) contribute about 15–25% of the sodium to an average diet (Fregley, 1981; Andres, 1982). In the processed form, such as sausage, these products frequently contain more than 300 mg of sodium per serving, and the American Medical Association suggests that products containing 300–500 mg of sodium per serving are the best candidates for sodium reduction (AMA, 1983).

However, reducing the sodium content in muscle foods to help decrease hypertension is a gross oversimplification and many other factors must be considered. These include several nutritional and physiological aspects of hypertension as well as quality and safety implications for various muscle foods. A realistic balance between "necessary" and "excess" sodium intake is a major current concern to the processing industry, regulatory agencies, medical profession, and consumers.

DIETARY SODIUM AND HYPERTENSION

Sodium is an essential nutrient that is not stored by the body when consumption is high, and thus a certain minimum dietary intake is essential. About 200 mg of sodium per day (equivalent to 0.5 g of sodium chloride) is adequate to maintain physiological balance (Gardenswartz and Schrier, 1982), while average consumption by Americans is most frequently estimated at 10–12 g of sodium chloride daily (IFT, 1980; Kolari, 1980; Fregley, 1981) with individual variation as high as 18 g or more (Hansen and Wyse, 1980). A diet with the equivalent of less than 3 g of sodium chloride per day is difficult to attain (Tobian, 1979) and most recommendations are for 3–8 g per day (NAS, 1980; Fregley, 1981). However, Freis (1976) estimated that an intake of 1–3 g per day may be the level at which the incidence of hypertension begins to increase. Relatively little further increase in blood pressure occurs with consumption of 5–15 g of sodium chloride per day. Other authors have questioned the effectiveness of sodium reduction in a typical diet (Kolata, 1982). Abernethy (1979) pointed out that the response curve of blood pressure with dietary sodium may be relatively "flat" over the range of most diets (5–15 g per day) with the most marked changes in blood pressure occurring at very low (1–3 g per day) or very high (about 25 g per day) sodium chloride levels.

The physiological role of sodium is to provide the proper osmotic balance in the extracellular fluid of the body. Extracellular sodium and intracellular potassium create the cell membrane environment that is necessary for nerve impulses, muscle function, and cellular utilization of many metabolic components. Transport of amino acids and sugars, for example, in many tissues are dependent upon sodium gradients (Gardenswartz and Schrier, 1982). Perhaps one of the most obvious effects of sodium, however, is its influence on extracellular fluid volume (ECFV); a function that has led to the implication of sodium in the etiology of hypertension. The sodium concentration in the extracellular fluid is strictly controlled by several renal, neural, and hormonal mechanisms. This might be expected from the importance of the cell membrane gradient in homeostasis. To maintain proper sodium concentrations, physiological adjustments to sodium fluctuations are usually made by adjusting the fluid volume. Meneely and Battarbee (1976), for example, estimated that retention of 20 meq of sodium per day would increase ECFV about 1 L per week. As a result, in normal individuals, an increased sodium consumption causes an expansion of the ECFV and an increase in body weight due to water retention. If dietary sodium is then decreased, diuresis and weight loss occurs until a "steady state" is again achieved (Gardenswartz and Schrier, 1982).

Since the Western diet is characterized by a high sodium intake, the body has had to adjust to a consistently high sodium load. In about 20% of the U.S. population which is genetically predisposed, this has apparently led to "essential" hypertension (Tobian, 1979), a term used to describe the 90% of hypertension cases for which the cause is unknown. The incidence of hypertension in some groups such as the black population may be as high as 40% (AMA, 1983) and the estimate of affected individuals in the United States

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approaches a total of 40 million people. Further, hypertension becomes increasingly prevalent with age (White and Crocco, 1980). The significance of this physiological condition is the increased probability of cardiovascular disease with increased blood pressure. Abernethy (1979) ranked hypertension first as a risk factor for cardiovascular disease and indicated that a diagnosis of hypertension doubles the risk of death from stroke, heart attack, or kidney disease. Data from the insurance industry has demonstrated increased mortality with increased blood pressure without any clear line of division between favorable and unfavorable blood pressure values (Paul, 1977).

Hypertension is clearly a health concern, and while the sequence of events leading to hypertension can be described, the underlying cause has been difficult to determine and at this point has not been clearly identified. The sequence of events that results in hypertension begins with an increased ECFV. Cardiac output increases as a result and blood pressure rises. In a normotensive individual, peripheral vascular resistance to blood flow then decreases to allow cardiac output to decrease to normal while the fluid volume is being reduced by the kidney (Mark et al., 1981; Rapp, 1982). In essential hypertension, however, the peripheral resistance increases and remains high, even after cardiac output and fluid volume return to normal. Consequently, blood pressure remains elevated (Frohlich and Messerli, 1982). Though it appears that there may be multiple causes of essential hypertension, all appear to result in a reduced ratio of renal excretory capacity to water and electrolyte intake (Guyton, 1977). As a result, blood pressure remains high, probably to increase excretory capacity.

Dietary sodium has been implicated as a causative agent in essential hypertension as a result of work in three general areas. First, there have been numerous epidemiological studies documenting little or no hypertension in primitive societies where little or no salt is used (Oliver, et al., 1975; Freis, 1976; Page, 1976; Abernethy, 1979; White and Crocco, 1980). Further, when these primitive societies have been introduced to salt, the incidence of hypertension almost invariably increases. Despite many other potential contributing factors, the most frequently identified common denominator has been salt (sodium) consumption. While a relationship of sodium intake to hypertension can be found in a comparison of different societies, the relationship does not usually hold for comparisons within a society (Staessen et al., 1981). Hypertension in an individual cannot be predicted by the level of sodium consumption (Tobian, 1979). Consequently, even though there is strong epidemiologic evidence relating sodium to hypertension, there is also equally strong evidence that the relationship is not a simple one.

The second area of work implicating sodium in hypertension has been clinical case histories where therapeutic diets very low in sodium have been successfully utilized to alleviate high blood pressure. This area has been reviewed (Dahl, 1977; Tobian, 1979), and, again, the relationship is not a simple one. Dietary sodium restriction reduces blood pressure in some individuals but not others, just as increased dietary sodium increases blood pressure in some but not others (Meneely and Battarbee, 1976). Only about 10–30% of the hypertensives on sodium restricted diets are likely to experience reduced blood pressure (Andres, 1982). In addition, body weight reduction can often be used to reduce hypertension independent of sodium intake (Reisen et al., 1978; Altschul and Grommet, 1980), and both sodium restriction and weight control are routinely prescribed for people with hypertension (Filer, 1980).

The third line of evidence linking sodium to hypertension is derived from experimental animals (particularly rats) which show hypertensive response to elevated dietary sodium chloride (Dahl, 1972). This work has been especially valuable because it clearly demonstrated a genetic factor in hypertension. The genetic factor has helped explain many of the discrepancies observed in epidemiological and clinical studies and has allowed the development of "salt-susceptible" and "salt-resistant" strains of rats (Dahl, 1977) for subsequent physiological comparisons (Rapp, 1982). One investigator (Dahl, 1977) has studied more than 45,000 rats over a 25-year period and has demonstrated various dose-response curves of sodium intake versus blood pressure in susceptible rats. On the other hand, Dahl found that the resistant rats can consume relatively high sodium levels without increased blood pressure. Tobian (1979) has suggested that human beings are also genetically susceptible or resistant to the development of hypertension. Feinleib et al. (1980) using twins, estimated that blood pressure in humans was about one-half genetically related and one-half environmental. The genetic predisposition to hypertension is a latent characteristic which manifests itself in response to environmental conditions, one of which appears to be dietary sodium. There are several other factors that may be involved, some of which are demonstrated in Fig. 1. These will be discussed in turn.

PHYSIOLOGICAL MECHANISMS

The physiological response to elevated dietary sodium is redundant with control mechanisms, probably reflecting the importance of precise sodium-potassium regulation in the body. However, it has become clear that the initial response is centered in the kidney. An increased sodium intake results in an expanded ECFV in order to maintain osmotic balance. The resulting increase in blood pressure allows for greater renal filtration (pressure filtration) and removal of both sodium and water (Frohlich and Messerli, 1982) via the urine. With essential hypertension, the ECFV, cardiac output, and sodium concentration may return to near normal but increased vascular resistance (and blood pressure) remains, perhaps to maintain the necessary renal filtration efficiency. It has been suggested that in hypertensive individuals, the kidney has a higher pressure requirement or "set point" that must be reached in order to adequately remove excess sodium and water (Freis, 1976). It has been demonstrated that isolated kidneys from salt-susceptible rats excrete 52% less sodium than kidneys from resistant rats when both are perfused at the same pressure (Tobian et al., 1978).

There are several known mechanisms (Fig. 2) involved with the renal and vascular adjustments necessary to deal with elevated sodium and ECFV. One of the best known is the renin-angiotensin-aldosterone system. Renin is secreted by the kidney in response to sympathetic nervous stimulation and initiates a series of events

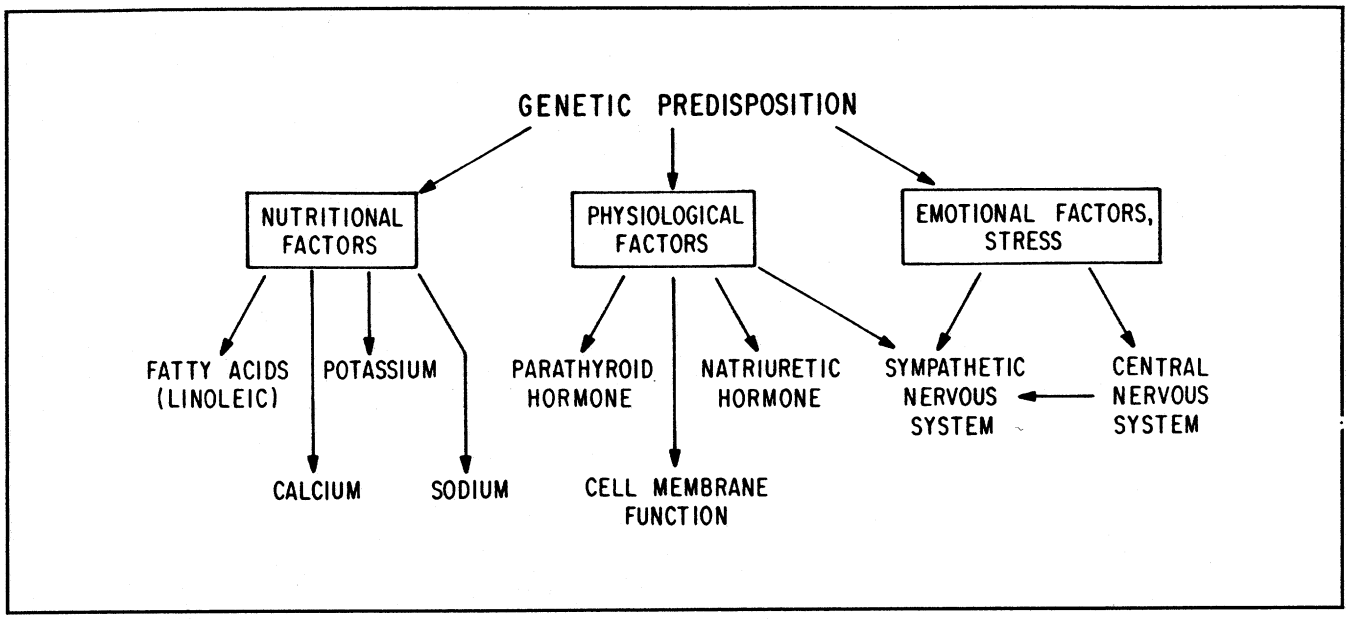
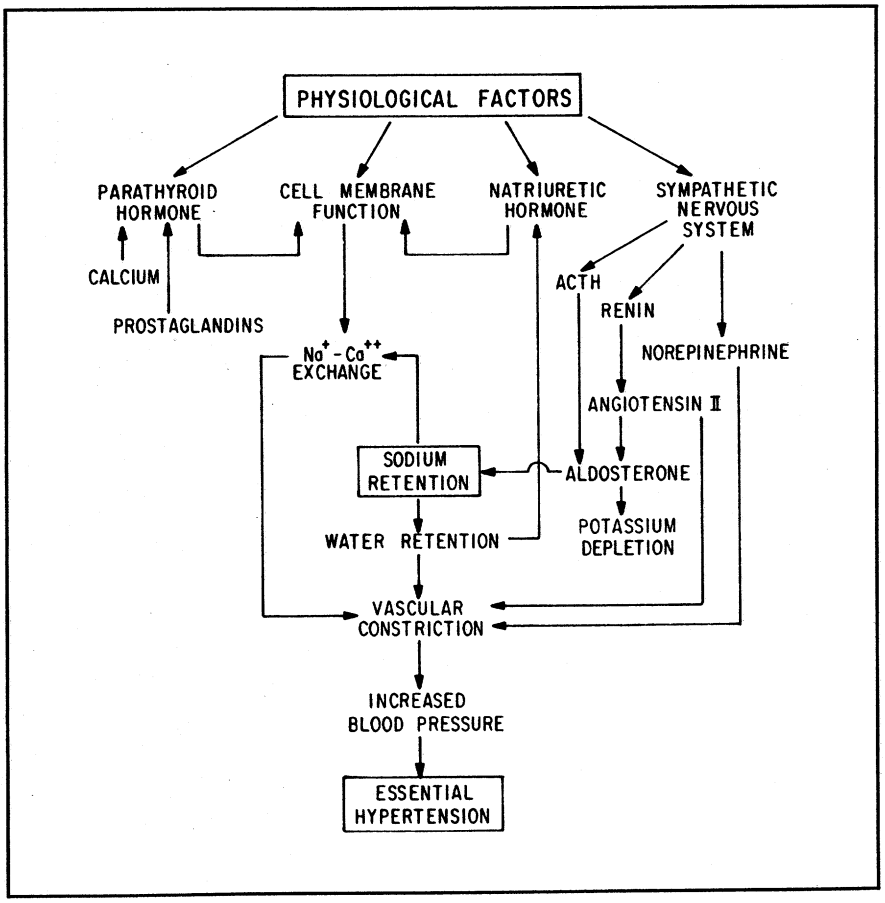


Fig. 1 (above)—**FACTORS THAT MAY BE INVOLVED IN THE DEVELOPMENT OF ESSENTIAL HYPERTENSION** *in genetically predisposed individuals*

Fig. 2 (right)—**SOME PHYSIOLOGICAL RELATIONSHIPS THAT MAY CONTRIBUTE TO HYPERTENSION**



resulting in angiotensin II which is a potent vasoconstrictor. The result is constriction of smooth muscle in the vascular system to increase peripheral resistance to flow and increase blood pressure. Renin-angiotensin II also stimulates the adrenal gland to release aldosterone, a hormone which acts on kidney tubules to increase reabsorption of sodium and elimination of potassium. This system could induce hypertension by either smooth muscle constriction or faulty sodium retention (Pearson and Wolzak, 1982). However, hypertensive individuals have been shown to have high, normal, or low renin activity and "high renin" versus "low renin" hypotheses have been advanced. High renin hypertension may be caused by the vasoconstriction induced by angiotensin II while low renin hypertension might result from a lack of adjustment to the increased blood volume induced by high sodium intake (Laragh, 1973).

The low renin hypothesis has always been somewhat unclear, however, and has led to the concept of a natriuretic hormone (Marx, 1981), a circulatory factor that is not yet clearly identified (Gardenswartz and Schrier, 1982). This proposed hormone may originate from the hyothalamus in response to blood volume increases and functions by reducing sodium reabsorption in the kidney tubule. The specific effect on kidney tubules is one of inhibiting the Na^+-K^+-dependent ATPase which generates energy for the sodium reabsorption "pump" (Marx, 1981), and this has significance for the potential development of hypertension. The decrease in sodium reabsorption should result in sodium excretion and decreased blood pressure, but apparently the natriuretic hormone inhibits the sodium pump in other cells as well,

including the smooth muscle cells of the vascular system. As a result, the sodium gradient across the smooth muscle cell membrane is lessened which may increase contractile behavior of smooth muscle. It has been suggested that the change is due to altered membrane exchange of sodium and calcium (Marx, 1981). Normal smooth muscle contraction is activated by an increase in intracellular cytoplasmic calcium which appears to be quite dependent on relatively large extracellular calcium stores (Casteels and Droogmans, 1982; van Breeman et al., 1982). Influx of calcium across the membranes may be an important part of smooth muscle regulation (Daniel et al., 1982), and the calcium gradient appears to be related to (perhaps set by) the sodium gradient. A sodium-calcium exchange (van Breemen et al., 1982) could mean that as intracellular sodium increases, so would intracellular calcium, a situation enhancing smooth muscle contractility and vasoconstriction (Blaustein, 1977). Therefore, drugs that reduce cellular calcium influx may be effective against hypertension by relaxing smooth muscle (Belizan et al., 1983). There is also speculation from data involving ionic exchange in red blood cells that membrane exchange rates may be genetically determined and that the sodium-calcium exchange in smooth muscle membranes may allow greater intracellular calcium in hypertensive individuals (Kolata, 1981). An abnormal calcium metabolism in hypertensive individuals has been suggested (McCarron et al., 1982).

Other hormonal systems that may be involved in hypertension include a variety of steroid compounds. Cortisol and hydroxycortisone as well as estrogens and androgens appear to increase sodium retention by the kidney, though none are as potent as aldosterone (Frohlich and Messerli, 1982).

Adrenal steroid secretion follows a different characteristic pattern in salt-susceptible or resistant rats and can be used to identify the two strains (Rapp, 1982).

Since a permanently increased peripheral resistance to blood flow is the hallmark of essential hypertension, vascular smooth muscle has been closely examined for structural changes and abnormalities. Hypertrophy, rigidity, and high mitochondrial calcium in hypertensive smooth muscle have been observed (Frohlich and Messerli, 1982; Somlyo et al., 1982). There is also a greater sensitivity in hypertensive smooth muscle to vasoconstrictors such as angiotensin II and catecholamines (Frohlich and Messerli, 1982). However, it is not yet clear whether there is a defect in smooth muscle that contributes to hypertension or if the changes observed reflect normal adjustments to the other physiological changes induced by hypertension.

Sympathetic nervous control also seems to play a role in essential hypertension since destruction of the sympathetic nervous system in susceptible rats on a high salt diet reduced the elevated blood pressure (Mark et al., 1981). Hypertension could result from a sodium-facilitated release of norepinephrine from adrenergic nerve endings resulting in stimulation of smooth muscle or from increased sympathetic innervation of blood vessels. β-endorphins will suppress the sympathetic nervous system and have been shown to reduce blood pressure (Kunos et al., 1981) providing further evidence of sympathetic nervous involvement with blood pressure regulation. The contribution of psychological stress to hypertension is probably mediated through the sympathetic nervous system (Light et al., 1983).

One other regulatory mechanism that responds to sodium is pituitary vasopressin (antidiuretic hormone) which increases water retention when sodium concentrations are high. However, this hormone does not seem to function abnormally in hypertension (Frohlich and Messerli, 1982; Rapp, 1982) and has not been considered to be a part of the problem.

Parathyroid hormone function is yet another physiological factor that may be involved through modification of membrane function. Since this may be induced by nutritional variables, it will be discussed later in that context.

The point to be made from considering the physiological changes in hypertension is that control of sodium levels and blood pressure is a complex interaction of many mechanisms, all of which are not yet completely understood with respect to hypertension.

NUTRITIONAL FACTORS

In addition to the physiological changes involving sodium control, there has been increasing evidence suggesting that several nutritional factors may play a major role in development of hypertension (Fig. 3). It has been known for some time that dietary potassium provides a protective effect against the hypertensive tendencies of sodium (Meneely and Battarbee, 1976; Abernethy, 1979; Lecos, 1983). Potassium supplementation generally decreases blood pressure and/or increases the life span for susceptible rats on high-sodium diets (Meneely, 1973). Similar blood pressure response to potassium has been noted in humans (Parfrey et al., 1981). Consequently, the dietary sodium/potassium ratio may be more important to hypertension than absolute sodium intake (Meneely and Battarbee, 1976). It's not clear how potassium contributes a protective effect, but the relationship is well enough established that dietary potassium recommendations in addition to sodium recommendations have been made (Abernethy, 1979; Hansen and Wyse, 1980; NAS, 1980; Lecos, 1983).

A relatively new area of nutritional research has developed around the potential role that calcium may play in hypertension. Geographic areas with water high in calcium and magnesium have been noted for decreased hypertension and calcium supplementation has been shown to decrease blood pressure in both rats and humans (McCarron et al., 1982). Belizan et al. (1983) found a 5–9% decrease in blood pressure of human subjects given 1 g of supplemental, calcium per day. A study of over 10,000 adults did not observe a high correlation between diet and blood pressure, but did find calcium most frequently as a dietary component correlated to blood pressure (Stanton et al., 1982).

The involvement of calcium may be due to the effects of parathyroid hormone which is released in response to low calcium levels. The parathyroid hormone, in attempting to conserve calcium, increases intracellular calcium which may again increase the responsiveness of vascular smooth muscle and increased peripheral vascular resistance to blood flow (Belizan et al., 1983). Calcium is also involved in prostaglandin synthesis which in turn inhibits parathyroid hormone release.

The involvement of prostaglandins can also be direct since they

enhance both vasodilations and renal sodium excretion (Henry and McCarron, 1982). Further, there is some speculation that the decreased blood pressure effect noted for dietary linoleic acid (Vergroesen et al., 1980) is due to increased prostaglandin synthesis originating from linoleic acid (Henry and McCarron, 1982). Vergroesen et al. (1980) indicated that an increased polyunsaturated/saturated fat ratio (P/S) in the diet may reduce blood pressure.

There are numerous other dietary factors beginning to come to light as potential contributors to hypertension (Henry and McCarron, 1982). Chloride may contribute to hypertension independently of sodium; while magnesium can provide a vasodilating effect and, when deficient, may enhance vascular sensitivity to vasopressors. Protein intake may increase calcium excretion and thereby increase hypertension. Other possible contributors include alcohol, trace minerals, and vitamins (Henry and McCarron, 1982). None of these relationships are well understood. As in the case of physiological changes brought about by sodium, a close examination of the nutritional factors contributing to hypertension show an increasingly complex relationship.

While reducing sodium intake has merit, it must not be done carelessly. For example, avoiding sodium frequently involves reducing consumption of dairy products with a corresponding decrease in dietary calcium. This might intensify hypertension rather than accomplish relief. Avoiding meat in order to reduce dietary sodium could result in iron or zinc deficiencies. Consequently, it would not be desirable to attempt to reduce dietary sodium by eliminating important food groups from the diet. A knowledgeable reduction of sodium, however, would reduce the current physiological excess being consumed and decrease the risk of hypertension in those individuals that are genetically predisposed. This might be of value to borderline hypertensive individuals who may not even be aware of their potential for hypertension. There are significant changes already underway in the food industry. Salt has been eliminated from infant foods since 1977 (Filer, 1980) since it was deemed unnecessary in those products and there are a large number of food processors who are currently reducing the sodium content in a variety of products (Food Chem. News, 1982a).

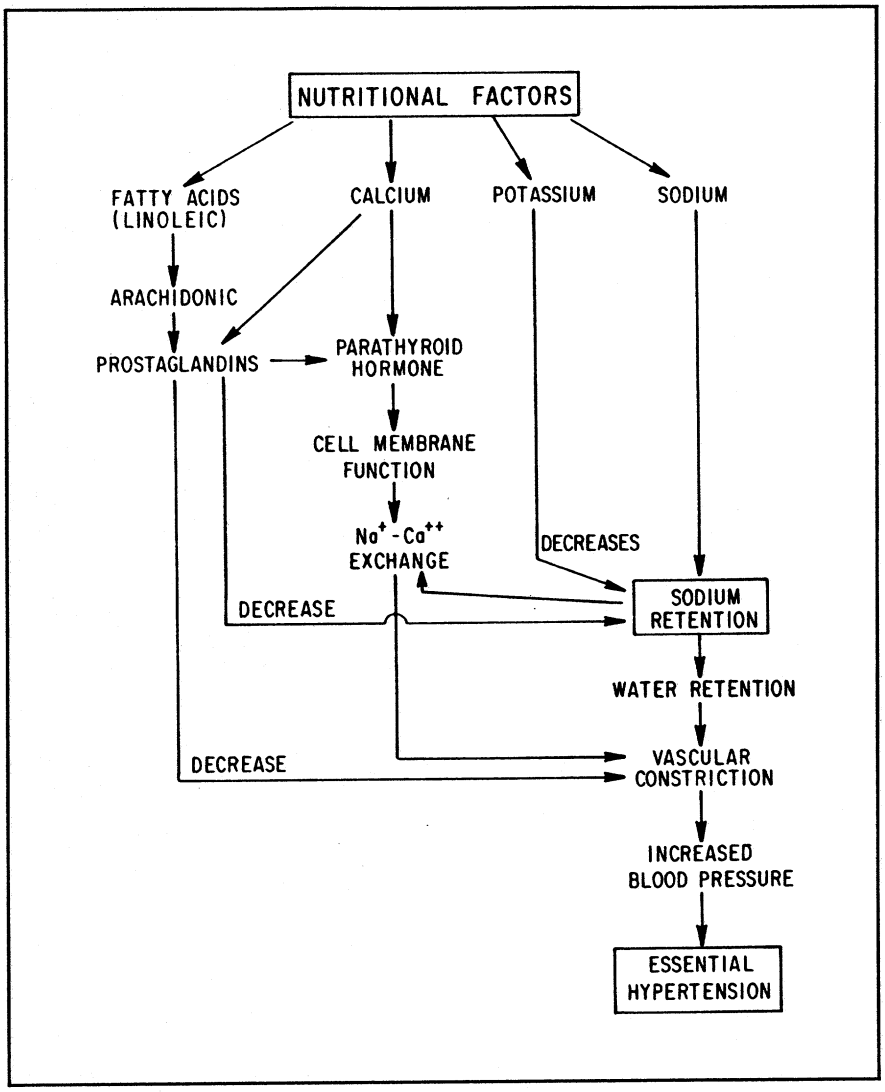


Fig. 3—**POTENTIAL ROLE OF SOME NUTRITIONAL FACTORS IN HYPERTENSION**

REDUCTION OF SODIUM IN MUSCLE FOODS

Any movement toward sodium reduction will involve muscle food products since they are significant sodium sources. Sodium content in muscle is significant in itself (ca 70 mg/100g), but the use of salt during processing increases the sodium to relatively high levels. The sodium content of various muscle foods reported by Marsh et al. (1980) is shown in Table 1 (fish), Table 2 (poultry), and Table 3 (red meat). It is obvious that processing or preparation techniques result in a product with greatly enhanced sodium concentration. It is also evident from USDA monitoring data (Food Chem. News, 1982b) that market products show a considerable variation in sodium content even within similar product categories (Table 4). The mean values for sodium in most of the products in Table 4 are similar to those reported by Marsh et al. (1980) but the range of sodium values shows a difference of 2–7 fold within product categories. These data also demonstrate that some products within each group are being marketed with relatively low sodium levels.

Muscle food processing involves several additives that contribute sodium (Marsden, 1980) but salt (sodium chloride) is the only one used in large enough quantities to offer opportunity for a significant reduction of sodium content. The meat industry is currently the largest user of salt in the food processing industry (Andres, 1982) while fish and marine products rank fourth. Since most of the sodium in processed products is controlled (added deliberately

during processing), a decreased sodium content can be achieved relatively easily.

Decreasing salt content, however, has many implications for muscle food products. Considerations to be made include textural changes, flavor differences, decreased moisture retention (yield), shortened shelf life (spoilage microorganisms), safety (pathogenic microorganisms), and even product appearance. The functional aspects of salt in processed meats have been addressed in earlier reviews (Marsden, 1980; Olson and Terrell, 1981; Terrell and Olson, 1981), and there are technological alternatives that allow some reduction of salt, without a great deal of product change. Use of phosphates, pre-blends, and pre-emulsified fats can compensate for some salt in terms of texture, binding ability, and yields (Rust and Olson, 1982). Use of a higher quality meat source as raw material, while more expensive, can also compensate for some salt reduction. Flavor changes will occur with less salt but, up to a point, may not be objectionable and may even be preferred by consumers. A gradual reduction of salt use by the general population, beginning with the elimination of salt in infant foods (Filer, 1980), may lead to a population with taste preferences for less salt (Kare and Brand, 1980). It is well established that preference for sodium chloride is an acquired taste and can be modified. Partial substitution of potassium chloride and other chloride salts for sodium chloride in processed meats has had some success (Seman et al., 1980, Whiting and Jenkins, 1981), but flavor differences, particularly bitterness, have been a limiting factor. Substitutes for table salt which are largely a mixture of potassium and sodium chloride have been successful in the consumer market (Anon., 1983c) but have not been well accepted for use in meat processing. Reformulation of other ingredients in order to maintain a desirable overall flavor when salt is reduced seems to be the route taken by at least one processor (Anon., 1981). The Homestead Provision Company appears to have been the first meat processor to produce a reduced-sodium product line, and they have received considerable attention for their products which have a 33.7% sodium reduction. Significant sales increases (Andres, 1982) in spite of a somewhat higher product cost (Anon., 1982) have been reported.

The reduction of sodium by 33.7% in the Homestead products is in line with various researchers who have suggested that sodium (as salt) reduction in appropriate products could range from 20%-40% depending on the product and conditions under which it is being manufactured (Seman et al., 1980; Whiting and Jenkins, 1981; Olson, 1982).

The implications of decreased shelf life and product safety, as sodium is reduced, is presently an area of major concern. The use of salt controls the microbiological

Table 1—**SODIUM CONTENT OF FISH AND SHELLFISH**[a]

Product	Sodium content (mg/100 g)
Raw Fish	
Catfish	59
Mackerel	47
Snapper	66
Lake trout	79
Cooked or Processed Fish	
Haddock (breaded, fried)	176
Flounder (baked with butter)	236
Herring (smoked)	6,158
Ocean perch (fried)	151
Salmon (canned, pink)	521
Tuna spread	329
Raw Shellfish	
Oysters	133
Scallops	255
Shrimp	161
Mussels	286
Cooked Shellfish	
Lobster (boiled)	249
Oysters (fried)	205
Scallops (steamed)	265
Shrimp (fried)	187

[a]Marsh et al., 1980.

Table 2—**SODIUM CONTENT OF POULTRY AND PROCESSED POULTRY PRODUCTS**[a]

Product	Sodium content (mg/100 g)
Cooked	
Chicken (roasted breast with skin)	70
Chicken (roasted drumstick with skin)	90
Turkey (roasted breast with skin)	53
Turkey (roasted leg with skin)	80
Gizzard (simmered)	61
Liver (simmered)	57
Processed Products	
Chicken frankfurter	1,371
Canned chicken	503
Chicken spread	411
Turkey roll	593

[a]Marsh et al., 1980.

Table 3—**SODIUM CONTENT OF RED MEAT AND PROCESSED MEAT PRODUCTS**[a]

Product	Sodium content (mg/100 g)
Skeletal Meats—Cooked	
Beef	65
Lamb	68
Pork	69
Veal	81
Organ Meats	
Heart (beef, braised)	104
Kidney (beef, braised)	254
Liver (calf, fried)	118
Liver (pork, simmered)	50
Processed Products	
Bologna (beef)	1,000
Bratwurst (cooked)	564
Braunschweiger	1,157
Frankfurter	1,121
Ham and cheese loaf	1,361
Olive loaf	1,486
Lebanon bologna	1,267
Pepperoni	2,033
Vienna sausage	950

[a]Marsh et al., 1980.

Table 4—**VARIABILITY OF SODIUM CONTENT IN SOME PROCESSED MEAT PRODUCTS**[a]

Product category	Sodium content (mg/100g)		
	Mean	Range	Ratio (high:low)
Hams	1247	654-2004	3.06
Canned luncheon meats	1294	830-1643	1.98
Pumped bacon	684	302-1249	4.14
Meat and poultry			
Bologna	1044	708-1482	2.09
Fresh pork sausage	625	140-1009	7.02

[a]Food Chem. News, 1982b

profile of meat, and a decrease of salt will, in general, reduce the shelf life. The problem with determining "how much" is that shelf life will also reflect an individual processor in terms of sanitation, quality of raw materials, heat processing, packaging, and temperature control. Some general relationships can be discussed, however. Kraft (1983) reported on studies involving microbial analysis during storage at 7°C of bologna, ham, and bacon with reduced sodium chloride. In bologna, bacterial counts were somewhat higher in the raw mixtures containing reduced salt, but subsequent smoke and heat processing reduced the counts of all products to the point where shelf life was not greatly affected by salt level. In ham, products with 1.5% salt showed more growth in storage compared to 2.0% and 2.5%, but no obvious spoilage occurred. With bacon, 0.9% salt resulted in spoilage after 7 weeks while product with 1.2% and 1.5% did not. Kraft (1983) concluded that, in general, a 25% reduction of salt did not adversely alter the shelf life or microbial characteristics of the products evaluated. Terrell and Brown (1981) recommended a brine content in frankfurters of 4% or more for optimum shelf life. In a frankfurter with 55% moisture, this would be about 2.3% salt.

$$(\%\ \text{Brine} = \frac{\%\ \text{salt}}{\%\ \text{salt} + \%\ \text{water}} \times 100)$$

Pathogenic staphylococci, typically one of the more salt tolerant pathogens, was also studied by Kraft (1983) who found low counts at all salt levels in ham or bacon.

Whiting et al. (1983) inoculated frankfurter mixtures that contained 1.5%, 2.0%, or 2.5% salt with *Staphylococcus aureus* or *Clostridium sporogenes* and stored the finished product at 11°C. There was no difference between products with 2.5% and 2.0% salt for total plate counts while the product with 1.5% salt was slightly (less than 1 log) higher. Staphylococci growth was unrelated to salt content and *Clostridium sporogenes* did not grow, probably due to presence of nitrite and less than ideal temperature. The 40% reduction in sodium chloride did not appear to greatly increase potential pathogenic growth and temperature was emphasized as much more important than salt content. It is interesting to note that the limited number of "reduced sodium" commercial meat products currently being marketed represent a 33.7% reduction in one case and a 25–40% reduction in another (Food Chem. News, 1982a).

Some theoretical concerns for safety remain, especially with respect to *Clostridium botulinum,* since nitrite and salt, both inhibitory agents, are both being reduced in processed meats. According to Hauschild (1982) many processed meats have been slowly decreased in salt content from a brine concentration of 5.0–5.5% in the late 1960s to around 4.0%. *Clostridium botulinum* can be a greater concern at these salt levels since a clear inhibitory interaction between salt and nitrite has been shown (Roberts, 1973) in model systems. At a constant temperature and pH, decreased salt required increased nitrite to prevent growth. At pH 5.8, for example, each 1% decrease in salt required about 50 ppm more nitrite to attain the same degree of control. Hauschild (1982) estimated that with less than 4% brine in meat, 100 ppm or more nitrite was needed. Eklund (1982) indicated that, in fish, the addition of 100 ppm nitrite allowed a 0.5% to 1.3% reduction of salt while retaining the same *C. botulinum* inhibition. Foster (Anon., 1983a) has been reported to have found a linear relationship between salt and botulism toxin formation in frankfurters, with every 0.25% increase in salt giving two more days of storage without toxin formation.

It seems clear that there is an interaction between salt and nitrite which may be important to microbial control. In some products where both salt and nitrite are already low, changes should be made with care.

There are implications for product safety in other areas as well. A minimum salt content is required for destruction of trichina in pork where it is not controlled by other means. New information as well as regulatory change is needed before any reduction in salt can be considered for these products. There is currently a proposal (Federal Register, 1983) under consideration for regulatory approval of additional trichina control methods. Fermented products depend on salt for selective microbial growth during fermentation, and it is difficult to predict how the microbial profiles might change with reduction of salt. In all cases concerning product safety, it is well to remember that product variation is a fact of life and any one product may vary from batch to batch for pH, initial bacterial counts, nitrite levels, composition, etc. Consequently, a considerable degree of overkill or overprotection is necessary to compensate for those combinations of circumstances that could otherwise lead to a hazard. In addition, some product abuse during distribution may occur especially in terms of storage temperatures.

Muscle food products with reduced sodium content represent a potential new market which could grow substantially due to consumer interest. Sodium chloride in many products can be reduced up to a point; a point which will depend upon the status of all the other variables that determine product quality and safety. Processors are going to have to do their homework if consumer satisfaction and safety is to be maintained.

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